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PERIPHERAL CHANGES IN REGIONAL SWEATING RESPONSES TO
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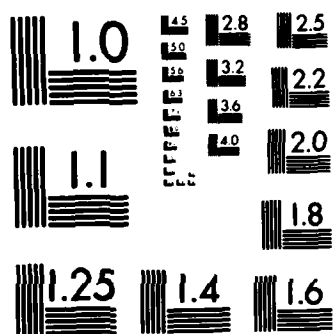
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intensity, altitude or environmental temperature. In all experimental conditions, the mean T_{es} threshold for the initiation of A (36.7°C) sweating was higher ($P < 0.05$) than C (36.5°C) or T (36.5°). The mean slopes of the $\dot{m}_s:T_{es}$ relationships for the three regional sites during the exercise-temperature combinations decreased with increasing altitude. Our data indicate that there are peripheral components active in the regional $\dot{m}_s:T_{es}$ relationship that occur in hypobaric hypoxia. Enhanced body cooling as a response to the higher evaporative capacity of the environment may be a component of these peripheral differences occurring in hypobaric hypoxia.

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PERIPHERAL CHANGES IN REGIONAL SWEATING RESPONSES TO
EXERCISE IN HYPOBARIC ENVIRONMENTS

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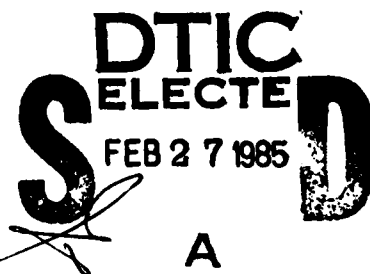
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ABSTRACT

The effect of hypobaric hypoxia on the relation of regional sweating to body temperature rise ($\dot{m}_s \cdot T_{es}$) was studied in four men and four women (follicular phase of menstrual cycle) who exercised at 40 and 60% of their altitude specific peak aerobic power at 770 Torr (sea level), 552 Torr (2596m), and 428 Torr (4575m) in 20°C or 30°C for 35 minutes. T_{es} and \dot{m}_s at the chest (C), arm (A) and thigh (T) were measured continuously from dew point sensors attached to the skin. No gender differences were found in either the sensitivity (slope) or the threshold of the $\dot{m}_s \cdot T_{es}$ for any site during any combination of exercise intensity, altitude or environmental temperature. In all experimental conditions, the mean T_{es} threshold for the initiation of A (36.7°C) sweating was higher ($P < 0.05$) than C (36.5°C) or T (36.5°C). The mean slopes of the $\dot{m}_s \cdot T_{es}$ relationships for the three regional sites during the exercise-temperature combinations decreased with increasing altitude. Our data indicate that there are peripheral components active in the regional $\dot{m}_s \cdot T_{es}$ relationship that occur in hypobaric hypoxia. Enhanced body cooling as a response to the higher evaporative capacity of the environment may be a component of these peripheral differences occurring in hypobaric hypoxia.

Key Words: Altitude, Esophageal Temperature, Dew-point, Local Sweating



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In hypobaric environments, the thermal capacity (the product of density and specific heat) of a unit volume of a gas mixture decreases under normal environmental temperatures. Theoretically, the same rate of total body sweat evaporation would cause a lower whole body skin wettedness ((ratio of evaporation from the skin (E_{sk}) to maximum evaporative capacity (E_{max})) at high altitude compared to sea level. The enhanced E_{max} of the hypobaric environment (16) is due to an increase in the mass transfer coefficient for a given effective air movement. The resultant effects on local sweating rate have not been studied to any extent.

In natives unacclimated to altitude, chronic altitude exposure (3800m) resulted in an upward threshold displacement of the core temperature for initiation of the total body sweat loss responses with no effect on the sensitivity of the sweating to core temperature relationship (17). During simulated altitude exposure (2000 and 4000m), it was concluded that whole body sweating was higher with increasing altitude when subjects exercised at the same absolute exercise intensity (7). To date, the relationship between local sweating and core temperature has not been investigated during acute altitude exposure.

In the present study, we examined the effect of acute moderate and high altitude exposure on the transient response of the local sweating (\dot{m}_s) to esophageal temperature (T_{es}) relationship. A modification of a central nervous system component would be indicated by an offset change in the esophageal temperature threshold for thermoregulatory sweating. Alternately, a peripheral modification (at the sweat gland site) would be demonstrated by thermal sensitivity (slope) changes in the relationship between \dot{m}_s : T_{es} for a given constant skin temperature.

METHODS

Eight subjects (4 men, 4 women) participated in the experiments after giving their informed consent. The subjects had an average (\pm SD) age of 26 ± 4 yr, weight of 71.1 ± 7.9 kg, and Dubois surface area of 1.82 ± 0.13 m². All subjects were accustomed to exercise and measurement routines. Female subjects were tested during the follicular phase (days 1-9) of the menstrual cycle to control for thermoregulatory differences that occur between the follicular and luteal phases (21). Subjects exercised in a semi-supine position behind a modified cycle ergometer. We determined peak aerobic power ($\dot{V}O_2$ peak) for each subject in this position as described previously (12). Separate maximal exercise tests were made at 770 Torr (sea level), 552 Torr (2596m), and 428 Torr (4575m) in a hypobaric chamber (ambient temperature = 24°C, dew-point temperature = 10°C). Oxygen consumption by standard open circuit techniques was measured on-line with a Hewlett-Packard (HP) computer modified for hypobaric environments. The $\dot{V}O_2$ peak was defined as the maximal oxygen uptake which occurred when increases in workload did not produce a subsequent increase in $\dot{V}O_2$. The mean (\pm SD) sea level $\dot{V}O_2$ peak was $2.92 (.68)$ l·min⁻¹, $2.68 (.56)$ l·min⁻¹ for 552 Torr, and $2.36 (.46)$ l·min⁻¹ at 428 Torr. The order of sea level and altitude $\dot{V}O_2$ peak tests was randomized, and subjects were not informed of the altitude during testing.

Experimental Procedures

Subjects reported to the hypobaric chamber between 0800 and 1200h after a light breakfast. Each subject was tested at the same time of day for all exposures to control for the circadian variation in heat loss responses. (20). They were dressed in shorts, socks, shoes and 100% cotton surgical scrub shirts. Submaximal exercise was performed by each subject a total of twelve times in

the semi-supine position at 40 and 60% of their individual altitude specific $\dot{V}O_2$ peak at sea level, 552 Torr and 428 Torr at two environmental temperatures (20°C and 30°C; $T_{dp} = 10^\circ\text{C}$). The exercise intensities were 47 ± 15 and 101 ± 30 W at sea level, 44 ± 10 and 95 ± 21 W at 552 Torr, and 40 ± 10 and 84 ± 18 W at 428 Torr. The order of conditions was randomized, and total time at any altitude during an experiment was less than 1.25h.

Subjects rested for 10 minutes after preparation and equilibrated to the specific environmental temperature before 35 minutes of semi-supine cycle exercise at the prescribed altitude and exercise intensity. Continuous measurements of esophageal (T_{es}), mean weighted skin temperature (\bar{T}_{sk}) from eight sites (15), and local sweating rate (\dot{m}_s) from the upper arm, chest and thigh (6) were recorded on a HP85 computer. Oxygen consumption was evaluated at rest and during steady-state exercise (last 15 minutes). Total body sweating rate was evaluated from changes in body weight corrected for convective and evaporative heat loss from the respiratory tract (5,9).

Statistical Analysis

The period of the rapid increase in \dot{m}_s was determined, and a regression equation of the $\dot{m}_s:T_{es}$ relationship was calculated for each subject during each experimental treatment. The T_{es} threshold for the initiation of sweating (i.e., evaporation above that owing to skin diffusion) was determined as the temperature when \dot{m}_s increased above $0.05 \text{ mg}\cdot\text{cm}^{-2}\cdot\text{min}^{-1}$. This was the T_{es} at which the regression line intersected the \dot{m}_s of $0.05 \text{ mg}\cdot\text{cm}^{-2}\cdot\text{min}^{-1}$.

A four-way analysis of variance was performed (subject by temperature by altitude by exercise intensity) using the slopes and thresholds of the $\dot{m}_s:T_{es}$ relationships. Post-hoc tests (Tukey) were performed whenever a significant F

ratio appeared ($P < 0.05$). Data in the Results section are presented as means \pm SD.

RESULTS

No gender differences were found in either the sensitivity (slope) or the threshold of the $\dot{m}_s:T_{es}$ relationship for any skin site during any combination of exercise intensity, altitude and environmental temperature. Slope and T_{es} onset data for chest sweating are shown in Tables 1 and 2 for the male and female subjects. There was no discernable transient period that could be analyzed for either slope or T_{es} threshold in the 40%/20°C (% $\dot{V}O_2$ peak/ T_a) condition due to the low sweating response resulting from the low core and skin temperatures in those experiments. These data will not be presented.

In all cases, the T_{es} threshold for arm sweating was higher (mean $\approx 0.17^\circ\text{C}$, $P < 0.05$) than either that for the chest or the thigh, and there was a lower ($P < 0.05$) slope for the chest compared to the arm and thigh (Tables 3 and 4). The T_{es} threshold for regulatory sweating was higher ($P < 0.05$) during the 60%/20°C condition than 60%/30°C and 40%/30°C at all altitudes, and the slope of the relationships ($\dot{m}_s:T_{es}$) for the 60%/20°C treatment at all altitudes was lower ($P < 0.05$) than for 40%/30°C (Tables 3 and 4).

The slopes (thermal sensitivity) of $\dot{m}_s:T_{es}$ for all exercise and environmental conditions for sea level, moderate and high altitude are shown in Table 3 for the three sites measured. These values are the mean of the eight subjects and illustrate the decreased sensitivity of the $\dot{m}_s:T_{es}$ relationship evident at increased altitude. Sensitivity of chest sweating was depressed an average of 33% ($P < 0.05$) at moderate altitude and 42% ($P < 0.05$) at high

altitude. Arm and thigh sensitivity were reduced an average of 43 and 23% ($P < 0.05$) respectively at moderate altitude and 44 and 30% ($P < 0.05$) at high altitude. This observation is more clearly demonstrated in Figure 1 in which the chest $\dot{m}_s:T_{es}$ relationship during the 60%/30°C treatment is compared across altitudes for a representative subject. In general, the suppression of the slope of $\dot{m}_s:T_{es}$ relationship with increasing altitude was not affected by the site of sweating measurement (Table 3). There was no significant effect of decreased barometric pressure on the T_{es} thresholds for the onset of regulatory sweating for any site where measured (Table 4). Whole body sweating rates during exercise were not different at sea level, moderate, and high altitude (Table 5). However, whole body sweating was greater at 60%/30°C exercise than the other two combinations. This was consistent across all altitudes.

Mean skin temperature did not differ with increasing altitude for any given combination of exercise intensity and environmental temperature (Table 6). Local skin temperature ($T_{s,l}$) for the three skin locations near the dew-point sensors are shown in Table 7. The only differences seen in $T_{s,l}$ are those resulting from the different constant ambient temperatures, and no effect of altitude is evident (Table 7).

DISCUSSION

The lowered air density with increasing altitude would theoretically result in greater skin diffusion and greater evaporation, therefore we controlled the workload at each altitude so that core temperature would rise to a similar level at each altitude (Table 8) allowing us to compare directly the effect of altitude on peripheral sweating due to the normal decrease in $\dot{V}O_2$ peak with increasing altitude. This investigation demonstrates a suppression in the sensitivity of the

relationship of local sweating rate to core temperature at both moderate and high altitude. This suppression in sweating sensitivity was not statistically different at 428 Torr and 552 Torr.

Greenleaf and colleagues (7) demonstrated an increase in whole body sweat loss at altitude in subjects exercising at the same absolute exercise intensity as sea level. However, these subjects exercised at a much higher percentage of their altitude-specific (4000m) $\dot{V}O_2$ max (approximately 65% compared to 45% at sea level), which possibly contributed to a larger demand for heat loss by evaporation and skin blood flow. In the present study, the total water loss during acute exercise was unchanged at both moderate and high altitude due to consistent relative work intensities across altitude. This earlier study (7) did not allude to depressed sensitivity at altitude perhaps due to the use of absolute versus relative exercise intensity and owing to the fact that the continuous measurement of local sweating is a more precise technique compared to body weight changes for evaluation of thermoregulatory control of sweating responses.

No upward shift in the core temperature threshold for sweating onset was observed in the present study. Thus, our results are not consistent with any central nervous system alteration in the control of sweating during exercise in acute hypobaric hypoxic exposure. Such a shift was described during exercise after chronic altitude exposure (17). However, the subjects in the previous study may have been dehydrated as a result of the chronic altitude exposure (11) which could account for the upward threshold displacement or delay in heat dissipation mechanisms previously demonstrated in dehydrated rabbits (23).

In our study the onset of arm sweating was delayed as compared to more centrally located sites, which confirms earlier observations during cycle ergometer exercise (13). The temperature of the arm ($T_{s,a}$) adjacent to the dew point sensor (Table 7) was lower than at the chest or thigh, which would account for the delayed onset of sweating (1,2,4,13). Arm sweating was consistently delayed among individuals and this delay occurred at all altitudes and ambient temperatures.

The depressed sensitivity (gain) in the $\dot{m}_s:T_{es}$ relationship measured from the chest compared to the more peripheral locations evaluated in our study does not agree with other observations (14) in which a higher gain and a lower onset T_{es} temperature occurred in the chest $\dot{m}_s:T_{es}$ relationship. However, subjects in the present study wore surgical scrub shirts and were seated in a chair behind the ergometer, thereby creating a different local environment at the chest, which may have influenced the local evaporative heat transfer at each site (5,3). The micro-environment at the chest (under a layer of cloth) had a higher water vapor pressure than at the thigh, which was manifested in the different local skin wettedness and heat transfer coefficients at the two sites. Local chest wettedness (0.72 at 4575m), measured with an unventilated dew point sensor, was approximately 40% higher than thigh (0.50) and arm (0.52) skin wettedness as determined by independent measurements of skin saturation pressure to ambient water pressure differences (10).

The sensitivity suppression of local sweating to esophageal temperature during acute exposure to moderate and high altitude appears to be a result of peripheral mechanisms. This modification may be caused by local skin influences, physical influences, and/or biochemical changes at the level of the sweat gland, or changes in the liquid-vapor interfaces within the skin itself. The local skin influences on the peripheral sweating mechanism are well documented

and have been discussed in great detail (1,2,4,13). In this study, mean skin as well as local skin temperatures were constant with increasing altitude, confirming that skin temperature is affected primarily by the ambient temperature (14,22), and is invariant with changes in altitude. We measured T_{sk} at both 20°C and 30°C and the T_{es} threshold for initiation of sweating was higher in the 20°C environment than the 30°C environment at each combination of exercise and altitude (Table 4); thus, ambient temperature affects $\dot{m}_s:T_{es}$ at moderate and high altitude in the same way as described for sea level environments (14). At altitude, the evaporative power of the environment is enhanced (5,16) with ensuing increased skin diffusion, which almost doubles at 5000m (3). Therefore, greater passive heat dissipation via insensible evaporative heat loss results in a depressed need for sweating. We have demonstrated at the same core temperature, a lower local sweating rate at moderate and high altitude of all skin sites examined, consistent across exercise intensity and ambient temperature indicating that these physical influences at altitude affect peripheral heat loss.

Following arterial occlusion, suppressed sweating is observed at a local skin site (4). It has been suggested that this effect is caused by a lower oxygen tension resulting in a decreased synthesis of transmitter substance, (i.e., acetylcholine). When physostigmine, an anticholinesterase, was administered in combination with arterial occlusion, the suppression of sweating did not occur. At an altitude of 4575m, arterial oxygen saturation is depressed, but probably not to levels induced by arterial occlusion. If local sweat gland activity was depressed solely by hypoxic influence, the gain of the local $\dot{m}_s:T_{es}$ relationship would be reduced, and core temperature would reach a higher steady-state level due to compromised evaporative heat loss. The results of our study do not

clearly support a local hypoxic inhibition of sweating; however, we cannot readily compare the environment of the individual sweat gland in the two studies except to the extent that arterial occlusion is a larger hypoxic stress than exercise in hypobaric hypoxia.

The present study has two major limitations: (a) the temperature of the skin area directly under the dew-point capsule was not measured, temperature was measured adjacent to the capsule; and (b) the skin temperature of the areas around the dew-point sensors was not maintained at a constant level, so as to completely standardize the local temperature impact upon the sweating response. Skin temperature decreases as evaporation occurs; however, during the periods of transient analysis, both mean skin and local skin temperature were constant with increasing altitude.

In summary, there were no changes in the esophageal temperature for the onset of regulatory sweating with acute moderate and high altitude exposure; however, depressed sensitivity of the local sweating to esophageal temperature relationship was apparent at both moderate and high altitude with no difference in suppression between altitudes. The data indicate that there are peripheral components in this relationship. These peripheral elements may be physical influences, biochemical changes at the sweat gland or changes in the liquid-vapor interfaces within the skin itself resulting in reduced input for thermoregulatory sweating. The difference in the physical environment at high altitude may adequately explain the thermoregulatory responses which were observed in this study, or there may be a direct hypoxic influence at the sweat gland, but this is less important in hypobaric hypoxia than during arterial occlusion as previously demonstrated (2).

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Table 1. Mean \pm (SD) slopes of chest $\dot{m}_s T_{es}$ for the male and female subjects at the combinations of altitude, temperature and exercise intensity combinations.

P_B	770 Torr	552 Torr	428 Torr
$\dot{V}O_2, T_a$			
60%, 20°C			
Males	.82(.36)	.55(.15)	.57(.23)
Females	.93(.19)	.72(.24)	.51(.09)
40%, 30°C			
Males	1.28(.88)	.83(.58)	.60(.28)
Females	1.25(.60)	.81(.24)	.74(.36)
60%, 20°C			
Males	1.00(.42)	.65(.26)	.62(.28)
Females	1.07(.46)	.69(.32)	.58(.20)

Table 2. Mean (\pm SD) esophageal temperatures for chest sweating for the male and female subjects at all altitude, temperature, and exercise intensity combinations.

P_B	770 Torr	552 Torr	428 Torr
$\dot{V}O_2, T_a$			
60%, 20°C			
Male	36.5(.2)	36.7(.4)	36.6(.3)
Female	36.8(.1)	36.8(.2)	36.7(.1)
40%, 30°C			
Male	36.4(.4)	36.4(.1)	36.5(.3)
Female	36.5(.2)	36.8(.1)	36.6(.1)
60%, 30°C			
Male	36.3(.5)	36.3(.4)	36.4(.4)
Female	36.7(.2)	36.6(.2)	36.4(.1)

Table 3. Mean (\pm SD) slope of the $\dot{m}_s:T_{es}$ relationship at sea level, moderate and high altitude for each site.

$\dot{V}O_2, T_a$	770 Torr			552 Torr			428 Torr		
	Chest	Arm	Thigh	Chest	Arm	Thigh	Chest	Arm	Thigh
60%, 20°C	0.87 (0.28)	1.08 (0.48)	0.98 (0.65)	.64 (0.21)	.60 (0.22)	.81 (0.20)	0.54 (0.19)	0.59 (0.24)	0.81 (0.41)
40%, 30°C	1.27 (0.69)	1.48 (1.21)	2.01 (1.25)	.82 (0.42)	.86 (0.49)	1.43 (0.34)	0.67 (0.29)	0.96 (0.42)	0.93 (0.26)
60%, 30°C	1.04 (0.40)	1.84 (0.67)	1.31 (0.41)	.67 (0.27)	1.07 (0.63)	1.01 (0.21)	0.60 (0.20)	0.90 (0.11)	0.95 (0.31)

Table 4. Mean (\pm SD) esophageal temperature for the onset of regulatory sweating ($\text{mg} \cdot \text{cm}^{-2} \cdot \text{min}^{-1}$) at sea level, moderate and high altitude for each site.

$\dot{V}\text{O}_2$, T_a	PB			770 Torr			552 Torr 428 Torr		
	Chest	Arm	Thigh	Chest	Arm	Thigh	Chest	Arm	Thigh
60%, 20°C	36.62 (0.21)	36.74 (0.22)	36.45 (0.24)	36.73 (0.29)	36.83 (0.24)	36.64 (0.27)	36.66 (0.21)	36.83 (0.23)	36.58 (0.10)
40%, 30°C	36.46 (0.28)	36.57 (0.23)	36.41 (0.16)	36.62 (0.22)	36.81 (0.20)	36.54 (0.22)	36.53 (0.20)	36.70 (0.14)	36.49 (0.24)
60%, 30°C	36.52 (0.45)	36.62 (0.30)	36.56 (0.38)	36.49 (0.33)	36.61 (0.37)	36.42 (0.31)	36.44 (0.25)	36.51 (0.27)	36.27 (0.25)

Table 5. Whole body sweating ($\text{g}\cdot\text{min}^{-1}$) for eight subjects at sea level, moderate and high altitude.

P_B	770 Torr	552 Torr	428 Torr
\dot{V}_{O_2}, T_a			
60%, 20°C	6.1 (2.0)	5.8(2.1)	6.5(1.8)
40%, 30°C	6.3(1.3)	6.0(1.3)	6.4(1.8)
60%, 30°C	10.1(2.5)	11.1(3.0)	9.4(1.9)

Values are mean \pm SD.

Table 6. Mean (\pm SD) skin temperature for the three combinations of exercise intensity and ambient temperature at the three altitudes during exercise transients.

		Sea Level			2696m			4575m		
		I	II	III	I	II	III	I	II	III
Mean		30.91	*34.46	34.65	31.34	*34.47	33.98	31.63	*34.63	34.08
SD		0.75	0.30	0.56	0.67	0.37	0.67	0.34	0.74	0.21
I is 60% $\dot{V}O_2$ peak, 20°C										
II is 40% $\dot{V}O_2$ peak, 30°C										
III is 60% $\dot{V}O_2$ peak, 30°C										

* lower ($p < 0.05$) than II and III.

Table 7. Mean (\pm SD) local skin temperature for the combinations of exercise intensity and ambient temperature at the three altitudes during exercise transients.

	<u>Sea Level</u>		<u>2693m</u>		<u>4575m</u>				
	<u>Chest</u>	<u>Arm</u> <u>Thigh</u>	<u>Chest</u>	<u>Arm</u> <u>Thigh</u>	<u>Chest</u>	<u>Arm</u> <u>Thigh</u>			
I	32.84 1.30	29.91 1.61	30.48 1.30	33.59 1.25	29.48 1.34	30.16 0.91	33.63 0.55	30.04 0.76	31.10 0.74
II	35.21 0.51	34.28 0.53	34.09 0.53	35.13 0.52	34.21 0.66	34.19 1.29	35.30 0.61	34.95 0.87	34.47 0.85
III	34.90 1.79	34.72 0.70	34.53 0.60	34.85 1.03	33.85 0.77	33.78 0.95	35.03 0.30	34.37 0.51	33.79 0.37

I is 60% $\dot{V}O_2$ peak, 20°C

II is 40% $\dot{V}O_2$ peak, 30°C

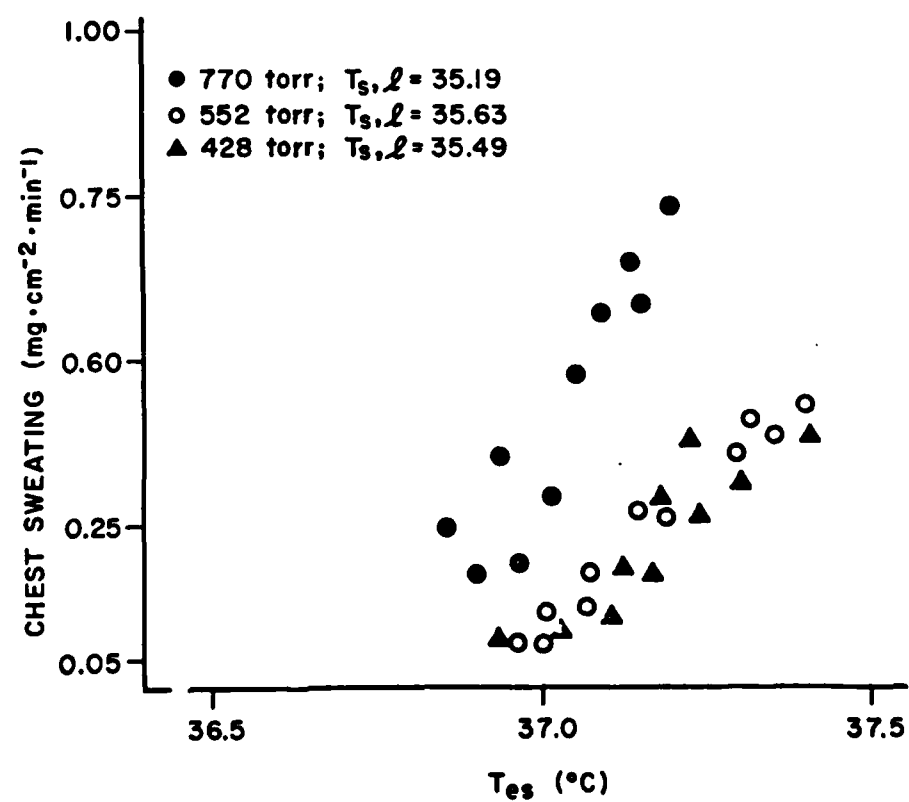
III is 60% $\dot{V}O_2$ peak, 30°C

Table 8. Mean (\pm SD) esophageal temperature at rest and during steady state exercise for the eight subjects.

<u>Rest</u>			
	<u>770 Torr</u>	<u>552 Torr</u>	<u>428 Torr</u>
\dot{V}_{O_2} , T_a			
20°C/60%	36.6(.2)	36.7(.3)	36.5(.2)
30°C/40%	36.7(.2)	36.6(.2)	36.5(.2)
30°C/60%	36.8(.2)	36.7(.2)	36.5(.2)
<u>Steady-State</u>			
	<u>770 Torr</u>	<u>552 Torr</u>	<u>428 Torr</u>
20°C/60%	37.1(.3)	37.2(.3)	37.1(.2)
30°C/40%	37.0(.2)	37.0(.2)	37.0(.2)
30°C/60%	37.3(.2)	37.3(.2)	37.1(.3)

FIGURE LEGENDS

Figure 1. Chest sweating plotted against esophageal temperature during 60% $\dot{V}O_2$ peak exercise at 30°C at three altitudes for a representative subject.



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